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Evaluating the triglyceride glucose index as a novel method for assessing insulin resistance in Turkish women with polycystic ovary syndrome

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ABSTRACT

Aims: Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder in women of reproductive age, characterized by insulin resistance (IR), hyperandrogenism, and polycystic ovary morphology. IR is a significant contributor to the pathogenesis and long-term complications of PCOS, but current gold-standard methods for assessing IR are often impractical for routine clinical use. This study aimed to evaluate the performance of the triglyceride glucose (TyG) index in identifying IR among Turkish women with PCOS and to assess its variability across different PCOS phenotypes.

Methods: This single-center, retrospective study included 247 patients diagnosed with PCOS according to the 2003 Rotterdam criteria. IR was assessed using both the TyG index and HOMA-IR. The study analyzed demographic and clinical data, including fasting plasma glucose, triglycerides, and various metabolic parameters. ROC curve analysis was used to determine the optimal TyG index cutoff for detecting IR.

Results: The mean age of participants was 24.09±5.53 years, with a mean BMI of 28.12±6.38 kg/m². The study identified a mean HOMA-IR of 3.46±1.82 and a mean TyG index of 4.51±0.26. A significant positive correlation was found between HOMA-IR and the TyG index ($r=0.370$, $p<0.001$). The optimal TyG index cutoff for detecting IR was 4.44, with a sensitivity of 70% and a specificity of 60% (AUC=0.693, $p=0.035$). The TyG index effectively identified IR across different PCOS phenotypes, though HOMA-IR revealed significant differences between some phenotypes.

Conclusion: This study is the first to demonstrate the effectiveness of the TyG index for predicting IR in Turkish women with PCOS and to explore its variability among phenotypes. The TyG index, based on fasting plasma glucose and triglyceride levels, offers a practical, cost-effective alternative to traditional methods for evaluating IR in PCOS.

Keywords: Triglyceride glucose index, insulin resistance, polycystic ovary syndrome, homeostatic model assessment of insulin resistance

INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder among women of reproductive age and the leading cause of anovulatory infertility.[1] It is marked by ovulatory dysfunction, hyperandrogenism, and polycystic ovary morphology (PCOM). PCOS is a multifaceted condition that arises in those who have a genetic susceptibility, influenced by environmental factors from the prenatal period onward. Changes in Gonadotropin-releasing hormone (GnRH) dynamics, resulting from the interaction of genetic and environmental factors, increase both the amplitude and frequency of luteinizing hormone (LH) pulses, as well as serum LH concentrations. Increased LH levels affect ovarian steroidogenesis, shifting it towards androgen production and resulting in a pause in follicle development. Additionally, insulin resistance (IR) and hyperinsulinemia contribute to increased ovarian androgen synthesis. The primary clinical findings of the disease are related to hyperandrogenemia (HA) and IR.[2] The diagnostic criteria for PCOS are established by three major groups; The National Institutes of Health/National Institute for Child Health and Human Diseases (NIH/NICHD) (1990), The European Society for Human Reproduction and Embryology/American Society for Reproductive Medicine (ESHRE/ASRM) (2003), The Androgen Excess-PCOS Association (2006).[3-5] PCOS is a lifelong disorder that can present with varying phenotypes. Even within the same patient, different phenotypes may appear at different times. Based on these diagnostic criteria, four distinct phenotypes were first defined and continue to be accepted today.[6] Phenotype frequency varies across different ethnic backgrounds. Additionally, the frequency can be influenced by whether the study population is drawn from clinic patients or the general community.

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IR is a cornerstone of both the pathogenesis and clinical findings of PCOS. Most women with PCOS display elevated levels of insulin, either at rest or in response to glucose, along with IR, regardless of body-mass index (BMI). IR has been observed in 50–70% of women with PCOS who maintain a normal BMI, and this percentage may be even greater among those who are obese.[7] The prevalence of prediabetes and type 2 diabetes mellitus (DM) among PCOS patients is reported to be 35–40%.[6] Each year, 2% of women with normoglycemia and 16% of women with prediabetes and PCOS progress to type 2 diabetes.[8,9] Therefore, PCOS is considered an independent risk factor for type 2 diabetes, and it is recommended that patients be regularly evaluated for glucose homeostasis.[6] Additionally, PCOS may be linked to various other metabolic conditions, including metabolic syndrome, dyslipidemia, and hepatic steatosis.[10] Therefore, early identification of IR and its variation across different phenotypes may be important. Although the hyperinsulinemic-euglycemic clamp (HIEC) is considered the gold standard for assessing IR, it is not suitable for clinical practice. Previous studies have shown that the homeostatic model assessment for IR (HOMA-IR) correlates with the HIEC.[11] However, measuring insulin levels is not typically included in routine examinations. The triglyceride glucose (TyG) index, calculated from fasting triglyceride (TG) and blood glucose (FBG) levels, offers a straightforward and cost-effective method for detecting IR. A systematic review concluded that the TyG index had a sensitivity of 96% and a specificity of 99% when the HIEC and HOMA-IR were used as reference tests.[12]

In our research, we sought to examine how effectively the TyG index identifies IR, and to evaluate its variation among different PCOS phenotypes.

METHODS

Ethics

The study was conducted with the permission of the Scientific Researches Evaluation and Ethics Committee of Ankara Etlik City Hospital (Date: 12.06.2024 Decision No: AESH-BADEK-2024-575). The study was conducted in accordance with the principles of the Helsinki Declaration.

Study Design

The study is a single-center, retrospective study conducted at the Endocrinology and Metabolism Department outpatient clinic of Ankara, in Turkey.

Demographic data and patients' clinical history were reviewed. We examined FPG, TG, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), follicle- stimulating hormone (FSH), LH, total testosterone (with LCMS), insulin at fasting status levels. The serum levels of LH, FSH, and insulin were assessed using chemiluminescence techniques (Siemens, New York, USA). Blood samples were collected from participants following an eight-hour fasting period during the follicular phase of menstrual cycle.

Calculated parameters related to IR: TyG index: ln[fasting Tg (mg/dl)×FPG (mg/dl)/2]; HOMA-IR: [FPG (mg/dl)]×[Fasting insulin (mIU/L)] / 405. IR was defined as a HOMA-IR value ≥2.5.

Study Participants

The study included adult patients with newly diagnosed PCOS who had not yet undergone any treatment related to the condition. Patients were diagnosed based on the 2003 Rotterdam criteria, which require at least two of the following three items: (I) oligo-ovulation and/or anovulation, (II) clinical and/or biochemical hyperandrogenism, and (III) polycystic ovaries.[4] Before diagnosing PCOS, PCOS-mimicking conditions were excluded. We assessed IR using both TyG index and HOMA-IR. We also examined differences in IR between PCOS phenotypes. We classified PCOS phenotypes based on the Rotterdam criteria as follows: A (oligo-ovulation, hyperandrogenism, polycystic ovaries), B (oligo-ovulation, hyperandrogenism), C (hyperandrogenism, polycystic ovaries), and D (oligo-ovulation, polycystic ovaries).

Exclusion Criteria

1. Women who had breastfed in last year
2. Pregnancy
3. Malignancy
4. DM
5. Using of medications that may affect glucose homeostasis
6. Non-euthyroid status

Patients who met the study criteria and agreed to participate were given detailed information about the study.

Statistical Analysis

The data were input into an Excel spreadsheet (Microsoft, Redmond, Washington) for analysis. Statistical analyses were performed using IBM SPSS Statistics software. To assess the normal distribution of the variables, we used both analytical methods (the Shapiro-Wilk test) and visual techniques, such as histograms. Normal continuous variables were expressed as means±standard deviations. Those with skewed distributions were expressed as medians with minimum and maximum values. To compare differences between two groups, we used either the independent sample Student's t-test or Mann-Whitney U test. To compare differences between three or more groups, we used one-way ANOVA. A significance level of 5% (type-I error) was used to assess statistical significance. Pearson correlation tests were conducted to investigate the relationships between variables and assess their significance.

RESULTS

The study included 247 patients from September 2022 to September 2024. The mean age of the patients was 24.09±5.53 [SD] years. Two (0.8%) patients with a diagnosis of primary hypothyroidism were receiving levothyroxine sodium treatment and were euthyroid. One (0.4%) patient had epilepsy, and one (0.4%) had accompanying depression. The mean BMI of the patients was 28.12±6.38 kg/m^2. Waist and hip measurements were available for 150 (60.7%) patients. The

mean waist measurement of these 150 patients was 89.24±14.48 cm, while the mean hip measurement was 106.12±15.57 cm. The mean waist/hip ratio was 0.84±0.07. Of the patients, 89 (36%) had obesity, 100 (40.5%) had a normal weight, and 58 (23.5%) were overweight (Table 1). One hundred thirty two (53.4%) patients were classified as phenotype A, 4 (1.6%) as phenotype B, 61 (24.7%) as phenotype C, and 50 (20.2%) as phenotype D.

Table 1. Baseline characteristics of the study participants

Patient number, n	**247**
Age (year)	24.09±5.53
BMI (kg/m²)	28.12±6.38
Waist circumference (cm, n=150)	89.24±14.48
Hip circumference (cm, n=150)	106.12±15.57
Waist/hip ratio	0.84±0.07
Weight classification, n (%)	100 (40.5%) normal weight 58 (23.5%) overweight 89 (36%) obese
Prediabetes, n (%)	21 (8.5%)
IR (according HOMA-IR), n (%)	163 (66%)
Dyslipidemia, n (%)	49 (19.8%)
Basal FSH (IU/L)	5.42±2.02
Basal LH (IU/L)	10.79±7.01
Basal total testosterone (ng/dl)	59±29.9
Fasting glucose (mg/dl)	85.41±9.37
Fasting insulin (mIU/L)	16.36±7.98
HOMA-IR	3.46±1.82
Total cholesterol (mg/dl)	177±33
LDL-C (mg/dl)	106±27
Triglyceride (mg/dl)	112±59
HDL-C (mg/dl)	51±13
TyG index	4.51±0.26

BMI: Body-mass index, IR: Insulin resistance, FSH: Follicle-stimulating hormone, LH: Luteinizing hormone, HOMA-IR: Homeostatic model assessment for IR, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, TyG: Triglyceride glucose

The mean values were as follows: FSH 5.42±2.02 IU/L, LH 10.79±7.01 IU/L, TT 59±29.9 ng/dl, and prolactin 15.79±12.91 ng/ml. The mean FPG was 85.41±9.37 mg/dl, while the mean fasting insulin level was 16.36±7.98 µU/ml. The mean HOMA-IR was 3.46±1.82. The mean values were as follows: total cholesterol 177±33 mg/dl, LDL-C 106±27 mg/dl, triglycerides 112±59 mg/dl, and HDL-C 51±13 mg/dl. The mean TyG index was 4.51±0.26. In repeated measurements, dyslipidemia was detected in 49 patients (19.8%), while prediabetes was detected in 21 patients (8.5%). Based on HOMA-IR, 163 (66%) patients had IR while 84 (34%) did not (Table 1). The distribution of baseline characteristics by phenotype is shown in Table 2.

When patients were categorized into normal weight, overweight, and obese groups, significant differences were observed in HOMA-IR and the TyG index (p<0.001, p<0.001). When normal weight and overweight patients were compared separately with obese patients, significant differences were found in both HOMA-IR and the TyG index (p<0.001 for all comparisons). However, when overweight patients were compared directly with obese patients, a significant difference was observed in HOMA-IR, but no difference was found in the TyG index (p<0.001, p=0.10). When comparing different phenotypes, a significant difference was observed in HOMA-IR (p<0.001), but no significant difference was found in the TyG index (p=0.87). When evaluating the phenotypes separately, a significant difference in HOMA-IR was found only between phenotypes A and C (p=0.024). There was no significant difference in age, BMI, waist/hip ratio, FSH, LH, TT, TK between phenotypes (p=0.95, p=0.18, p=0.27, p=0.32, p=0.65, p=0.78, p=0.17). There was no statistically significant difference in IR among PCOS phenotypes when assessed using the TyG index as a reference (p=0.86).

The correlation analysis revealed a significant positive relationship between HOMA-IR and the TyG index (p<0.001, r=0.37) (Figure). A significant positive correlation was found between the TyG index and BMI (p<0.001, r=0.36), the TyG index and waist/hip ratio (p<0.001, r=0.24), the TyG index and fasting insulin level (p<0.001, r=0.31), the TyG index and total cholesterol (TC) (p<0.001, r=0.55), and the TyG index and LDL-C (p<0.001, r=0.45). A significant negative correlation was also found between TyG index and HDL-C (p<0.001, r=-0.21). No statistically significant correlation was found between the TyG index and FSH, LH, or TT (p=0.06, p=0.11, p=0.72). The correlations of HOMA-IR and the TyG index with biochemical and anthropometric parameters are shown in Table 3.

ROC curve analysis identified an optimal cutoff value for the TyG index at 4.44, with a sensitivity of 0.70 and a specificity of 0.60 for identifying IR (AUC=0.693, p=0.035). All patients were categorized into two groups according to the TyG index cutoff values: group 1 (TyG index<4.44) and group 2 (TyG index≥4.44). We analyzed fasting IR-related metabolic parameters between the two groups (Table 4).

DISCUSSION

IR is a key factor in the pathogenesis and progression of long-term complications in individuals with PCOS. Hyperinsulinemia, in turn, plays a significant role in exacerbating hyperandrogenism and reproductive disorders. The dynamic euglycemic clamp technique, although recognized as the gold standard for measuring insulin sensitivity, is both costly and complicated. Moreover, it may not be available in all countries, making it less feasible for use in outpatient clinic settings. Therefore, there is a need to develop a simpler, more practical, and cost-effective method for assessing IR. Such a method would enable more accurate and personalized diagnosis, treatment, and prognosis for individuals with PCOS. Numerous studies have examined indirect tests, such as HOMA-IR for evaluating IR in PCOS.[13] Our study is among the first to investigate the effectiveness of the TyG index in assessing IR in PCOS patients within the Turkish population and to explore its variability across different phenotypes.

The TyG index has been recognized as an effective alternative to insulin testing for evaluating IR. This recommendation is supported by various studies that have highlighted the TyG index's effectiveness in evaluating IR within the general population.[12,14-16] Subsequently, studies have emerged demonstrating the utility of the TyG index for diagnosing IR in patients with PCOS. First study was published by Kwon et al.[17] and they evaluated 172 Korean PCOS patients. They reported a strong correlation between the TyG index and HOMA-IR (r=0.524). Their analysis identified an optimal

Table 2. Baseline characteristics according to phenotypes

	Phenotype A	Phenotype B	Phenotype C	Phenotype D
Patient number, n	132	4	61	50
Age (year)	24.15±5.80	23.50±3.31	23.79±5.33	24.34±5.30
BMI (kg/m²)	28.90±6.70	26.93±3.97	27.67±6.52	26.71±5.24
Waist circumference (cm)	89.14±14.31 (n=84)	N/A (n=0)	91.50±15.47 (n=38)	86.50±13.56 (n=28)
Hip circumference (cm)	106.01±13.45 (n=84)	N/A (n=0)	106.26±13.95 (n=38)	102.71±11.52 (n=28)
Waist/hip ratio	0.83±0.07	N/A (n=0)	0.85±0.09	0.83±0.06
Weight classification Normal weight, n (%) Overweight n (%) Obese n (%)	 47 (35.6) 32 (24.2) 53 (40.2)	 2 (50) 1 (25) 1 (25)	 28 (45.9) 12 (19.7) 21 (34.4)	 23 (46) 13 (26) 14 (28)
Prediabetes, n (%)	10 (7.5%)	3 (75.0%)	5 (8.1%)	3 (6.0%)
IR (According to HOMA-IR), n (%)	97 (73.5%)	4(100%)	34 (55.7%)	28 (56.0%)
Dyslipidemia, n (%)	28 (21.2%)	0	13 (21.3%)	8 (16.0%)
Basal FSH (IU/L)	5.36±2.20	4.50±1.60	5.28±1.85	5.85±1.72
Basal LH (IU/L)	10.55±6.87	14.74±7.03	10.63±7.73	11.25±6.51
Total testosterone (ng/dl)	58.06±29.80	56.53±8.75	62.39±24.90	57.54±34.46
Fasting glucose (mg/dl)	85.71±9.39	99.00±15.14	84.68±9.79	84.42±7.54
Fasting insulin (mIU/L)	50.18±12.52	65.50±27.24	51.33±10.95	55.52±16.08
HOMA-IR	3.69±1.80	5.60±1.83	3.06±1.78	3.18±1.73
Total cholesterol (mg/dl)	178.34±34.83	202.75±35.89	171.10±34.28	181.19±29.86
LDL-C (mg/dl)	109.12±28.06	128.75±33.39	99.39±28.02	107.97±24.20
Triglyceride (mg/dl)	113.93±60.52	100.25±42.57	112.47±64.86	107.46±52.56
HDL-C (mg/dl)	50.18±12.52	65.50±27.24	51.33±10.95	55.52±16.08
TyG index	4.52±0.25	4.56±0.17	4.50±0.29	4.50±0.22

N/A: not available

BMI: Body-mass index, IR: Insulin resistance, FSH: Follicle-stimulating hormone, LH: Luteinizing hormone, HOMA-IR: Homeostatic model assessment for IR, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, TyG: Triglyceride glucose

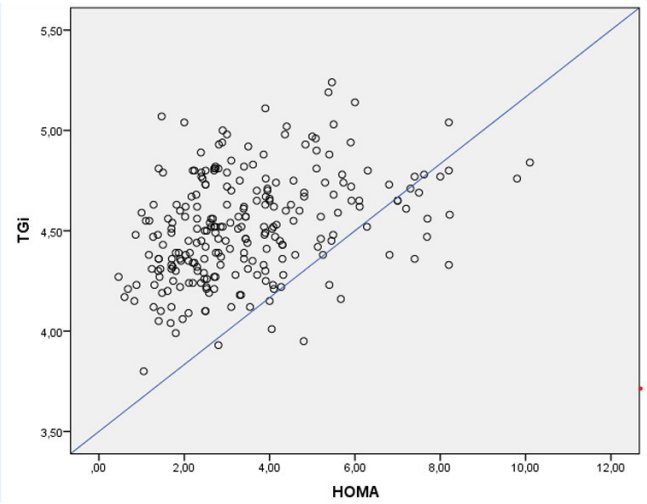

Figure. Correlations between TyG index and HOMA-IR
TyG: Triglyceride glucose, HOMA-IR: Homeostatic model assessment for insulin resistance

Table 3. Correlation analysis

	TyG index (p-value, r-value)	HOMA-IR (p-value, r-value)
BMI	p<0.001, r=0.362	p<0.001, r=0.546
Waist/hip ratio	p<0.001, r=0.248	p<0.001, r=0.273
Fasting plasma insulin	p<0.001, r=0.318	p<0.001, r=0.955
TC	p<0.001, r=0.550	p=0.09
LDL-C	p<0.001, r=0.455	p<0.001, r=0.201
HDL-C	p<0.001, r=0-0.219	p<0.001, r=-0.306
FSH	p=0.06	p<0.001, r=-0.171
LH	p=0.11	p=0.22
Total testosterone	p=0.72	p=0.26

Statistical significance was established when p<0.05, BMI: Body-mass index, TC: Total cholesterol, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, FSH: Follicle-stimulating hormone, LH: Luteinizing hormone

Table 4. Comparison of metabolic parameters between the two groups divided according to the cutoff value of triglyceride and glucose index

	Group 1 (IR-) (n=97)	Group 2 (IR+) (n=150)	p value
BMI (kg/m²)	25.49±5.39	29.82±6.41	p<0.001
Waist/hip ratio (cm)	0.82±0.08 (n=64)	0.84±0.07 (n=86)	p=0.13
Fasting glucose (mg/dl)	82.55±8.23	87.26±9.63	p<0.001
Fasting insulin (µU/ml)	13.43±6.77	18.24±8.15	p<0.001
HOMA-IR	2.74±1.44	3.93±1.89	p<0.001
Total cholesterol (mg/dl)	158.7198±26.65	189.43±32.77	p<0.001
LDL-C (mg/dl)	93.87±23.88	115.17±26.68	p<0.001
Triglyceride (mg/dl)	126.07±57.73	143.92±56.08	p<0.001
HDL-C (mg/dl)	62.74±13.83	52.36±10.65	p<0.001

Statistical significance was established when p<0.05. BMI: Body-mass index, IR: Insulin resistance, HOMA-IR: Homeostatic model assessment for IR, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol

TyG cutoff value of 8.126, with a sensitivity of 0.807 and a specificity of 0.683, for detecting IR. Similarly, our study also demonstrated a correlation between HOMA-IR and the TyG index (r=0.370). However, our study determined that the optimal cutoff value for the TyG index in detecting IR in PCOS patients was 4.44. In their study, the mean HOMA-IR was 2.28±2.21, whereas in our study, it was 3.46±1.82. The fact that IR was higher in our study population may have caused the difference. Kheirollahi et al.[18] investigated the TyG index in women with PCOS in Iran, IR was detected by HOMA-IR in 9.83% of the patients. In our study, this rate was found to be 66%. This discrepancy could be attributed to differences in patient characteristics, such as BMI, which was 26.62±4.19 kg/m² in their study compared to 28.12±6.38 kg/m² in ours. Additionally, their study used a HOMA-IR cutoff of ≥2.63,

which might also contribute to the variation in detection rates. Similarly, their study also demonstrated a correlation between HOMA-IR and the TyG index (r=0.233). They identified the optimal cutoff point for the TyG index in predicting IR as 4.65, with a sensitivity of 63% and a specificity of 60%. The variability in the correlation strength between the TyG index and HOMA-IR across studies may be attributed to differences in cutoff values and population characteristics. Studies utilizing higher TyG cutoff points, such as 8.126 and 8.51, demonstrated stronger correlations with HOMA-IR.[17,20] In contrast, the lower cutoff values in our study (4.44) and Kheirollahi et al's study (4.65) were associated with weaker correlations.[18] This discrepancy could stem from differences in fasting insulin levels (16.36 μU/mL in this study vs. 9.98 μU/mL in Kwon et al's study) and BMI scores (28.12 kg/m² in this study vs. 26.62 kg/m² in Iranian study).[17,18] Higher fasting insulin and BMI levels may result in elevated HOMA-IR scores, thereby weakening the correlation with the TyG index. Furthermore, ethnic variations, such as the lower BMI and IR typically observed in Far Eastern populations, could also influence these findings. These factors emphasize the importance of considering population-specific metabolic characteristics when evaluating the TyG index as a marker for IR.

In a retrospective cross-sectional study by Yang et al.[19] the correlation between the TyG index and metabolic syndrome (MS) was explored. MS was identified in 32.5% of the subjects with PCOS, and the study demonstrated a strong association between the TyG index and MS in these women. Obesity, hyperglycemia, and dyslipidemia were diagnosed in 33.8%, 20.9%, and 33.1% of women with PCOS, respectively. In our study, the rates were 36% for obesity, 8.5% for hyperglycemia, and 19.8% for dyslipidemia. They discovered that the TyG index was independently linked to risk factors for metabolic syndrome in women with PCOS, such as hyperglycemia, obesity, and dyslipidemia. Similarly, we found a significant positive correlation between the TyG index and BMI, waist/hip ratio fasting insulin level, TK and, LDL-C. Zheng et al.[20] published a study assessing whether the TyG index is superior to other indices for diagnosing IR in patients with PCOS. The TyG index achieved the highest area under the ROC curve for predicting IR in patients with PCOS, as determined by HOMA-IR, with a value of 0.781 (95% CI: 0.693–0.853, p<0.001). At a cutoff point of 8.51, the TyG index demonstrated a sensitivity of 63.2% and a specificity of 87.0%. We did not evaluate other lipid ratios in defining IR. However, in our study, the TyG index demonstrated comparable effectiveness to HOMA-IR in identifying IR in patients with PCOS.

Głuszak et al.[21] examined whether there are hormonal, biochemical and metabolic differences between PCOS phenotypes. In both their study and ours, phenotype A was the most common (60.2%, 53.4%). In both their study and ours, no significant differences were observed between the subtypes regarding age, weight, height, waist to hip ratio, and BMI. However, while their study found no difference in HOMA-IR among the groups, our study revealed no difference in the TyG index between phenotypes. In contrast, we did find a significant difference in HOMA-IR between phenotypes A and C (p=0.024). In the study by Pehlivanov et al.[22] phenotype A was the most common, occurring in 58.6% of the cases. In contrast to our findings, their study reported that groups A and B were more obese and had higher levels of IR. However, this difference may be attributed to the smaller sample size in their study.

Limitations

This study has several limitations, such as its retrospective design and reliance on existing patient records, which could introduce selection bias and limit the applicability of the findings. Prospective cohort studies with larger sample sizes and longitudinal follow-up are needed to assess the predictive value of these indices for long-term metabolic outcomes in PCOS.

CONCLUSION

In conclusion, this study is the first to illustrate the utility of the TyG index for predicting IR in Turkish women with PCOS and to explore its variability among different phenotypes. Given the significance of carbohydrate homeostasis in the pathogenesis and complications of PCOS, the TyG index—based on fasting plasma glucose and triglyceride levels—emerges as an effective, easy-to-use, and cost-efficient method for evaluating IR during follow-up.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was conducted with the permission of the Scientific Researches Evaluation and Ethics Committee of Ankara Etlik City Hospital (Date: 12.06.2024 Decision No: AESH-BADEK-2024-575).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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